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THE POSTCHOLECYSTECTOMY SYNDROME AND ITS TREATMENT*

RALPH COLP

Clinical Professor of Surgery, College of Physicians and Surgeons
Columbia University, New York

REFINEMENTS in the technique of cholecystography have aided substantially in the accurate diagnosis of gallbladder disease. The recent improvements in the pre- and postoperative preparation of patients, together with the standardization of the surgical technique, have so lowered the operative mortality that cholecystectomy is a relatively safe procedure. Unfortunately, the results revealed by follow-up examinations after operation, especially in the group of patients with noncalculous cholecystitis, are far from satisfactory.[1,2,3,4] The causes of failure following cholecystectomy are many and the complaints which ensue may be bizarre.[5] Frequently the removal of the gallbladder is followed by acute symptoms identical to those which existed prior to operation. This postoperative symptom complex so characteristic of gallbladder disease is called the postcholecystectomy syndrome.

The postcholecystectomy syndrome may resemble cholecystitis and cholelithiasis in all their varied clinical manifestations and it may simu-

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TABLE I

END RESULTS OF OPERATION UPON THE GALLBLADDER AND COMMON DUCT[7]

Condition or Operation	Cases Followed No.	Re-Operation No.	Well No.	Well After One Year No.	Well After Two Years No.	Total Well		Improved		Not Improved	
						No.	Per Cent	No.	Per Cent	No.	Per Cent
1. Cholecystectomy for Chronic Cholecystitis with Stones.	119	2	70	22	2	94	78.9	14	11.8	11	9.3
2. Cholecystectomy for Chronic Cholecystitis without Stones.	8	0	2	0	1	3	37.5	0	...	5	62.5
3. Cholecystostomy for Chronic Cholecystitis with Stones.	1	0	1	0	0	1	100	0	...	0	...
4. Cholecystectomy for Acute Cholecystitis with Stones.	75	5	48	11	2	61	81.3	4	5.3	10	13.3
5. Cholecystostomy for Acute Cholecystitis with Stones.	7	1	4	1	0	5	71.4	1	14.3	1	14.3
Total Operations on Gallbladder.	210	8	125	34	5	164	78.1	19	9.0	27	12.9
6. Choledochostomy with Operation on Gallbladder.	12	2	6	0	2	8	66.7	1	8 3	3	25.0
7. Choledocholithotomy with Operation on Gallbladder	31	7	18	4	3	25	80.6	2	6.5	4	12.9
Total Operations on Gallbladder and Common Duct.	43	9	24	4	5	33	76.5	3	7.2	7	16.3

late many of the physical findings. The acute symptoms sometimes occurring during the convalescent period and most frequently noted during the first two years may be heralded by agonizing colic confined to the upper half of the abdomen, often radiating to either shoulder or the interscapular region. These attacks are commonly accompanied by nausea and vomiting, and are often associated with transient periods of mild jaundice, pruritus, and occasionally intermittent fever and chills. Physical examination may reveal abdominal soreness and occasionally a tinge of icterus.

Hellström,[6] reporting one thousand and forty-one cases in which cholecystectomy had been done, stated that about 30 per cent of the patients complained of upper abdominal pain and distressing pressure. The symptoms were similar in nature to the colicky attacks described prior to cholecystectomy. In only nine of the cases were the episodes due to residual ductal calculi, and in a few of the patients the attacks terminated in an acute pancreatitis. However, in the majority of cases Hellström offered no explanation which would account for the occurrence of the postcholecystectomy colic.

Doubilet,[7] in the Surgical Clinic of The Mount Sinai Hospital, personally interviewed two hundred and fifty-three patients following operations for gallbladder disease and saw them at regular intervals during periods varying from one to seven years. The end results are summarized in Table I. Practically 40 per cent of all patients who were followed suffered from postoperative symptoms, either temporary or permanent. The majority of this group described the acute episodes as resembling those which had existed prior to operation and in some cases the attacks were frequent and severe enough to require sedation. The pains were of two main types. In the larger group, seventy-eight patients stated that the intense postprandial pain lasted from five minutes to two hours and occasionally radiated to the back. In a smaller group comprising twenty-four patients the pain which lasted from two to twenty-four hours radiated from the left upper quadrant to the shoulders. It was frequently accompanied by persistent abdominal soreness and in two cases an acute pancreatitis developed. In our experience these symptoms might follow the surgical removal of any gallbladder regardless of its pathologic lesions. The postcholecystectomy syndrome was most frequent in those cases characterized by definite colic prior to operation and in which functional disturbances were

demonstrated by cholecystography, but in which surgical exploration revealed the absence of pathologic lesions of the gallbladder or the presence of a noncalculous cholecystitis. It seemed less frequent in cases of calculous cholecystitis. The syndrome was relatively rare when a fibrosed functionless gallbladder and dilated common duct were present.

The etiology of the postcholecystectomy syndrome may usually be attributed to a dyskinesia of the sphincter mechanism. This sphincter spasm may be stimulated either by local causes or by intrabiliary factors such as recurrent or residual calculosis of the cystic or common bile duct, partial traumatic strictures of the choledochus, cholangitis or pancreatitis. In some cases the dyssynergia may be initiated by psychic disturbances or by glandular dyscrasias, and in others it may be the result of a spastic colon. The significance and importance of biliary dyskinesia has been thoroughly reviewed by Ivy, Goldman and Sandblom,[8,9] Hill,[10] and Bergh and Layne.[11] It will only be necessary here to consider certain fundamental anatomic, physiologic and pathologic data which are concerned in the mechanism and production of most cases of postcholecystectomy syndrome. The anatomy of the periampullary region of the duodenum has been extensively investigated by Letulle and Nathan-Larrier,[12] Giordano and Mann,[13] and others, and has recently been examined from an embryologic standpoint by Boyden and his associates.[14,15] (Fig. 1) The latter have identified longitudinal fasciculi which probably served to erect the papilla and aid in the ejection of bile, a sphincter of the pancreatic duct, a definite sphincter at the terminal end of the choledochus, and in addition a sphincter of the ampulla which was found in about one-sixth of their dissections. The contraction of this muscle, while it prevents the flow of secretions into the duodenum, converts the choledochus and the pancreatic duct into one canal so that bile may pass into the pancreatic duct and pancreatic secretions may pass into the common bile duct. The pathologic significance of this, which is extremely important, will be subsequently discussed. Not only is the sphincter of the common bile duct a distinct anatomic entity, but Bergh[16] has presented evidence to show that it acts independently of the duodenal musculature to which it is intimately connected. However, functionally the sphincter mechanism includes both the intrinsic muscles of the papilla and the associated muscles of the adjacent intestinal wall. It prevents the regurgitation of duodenal contents, aided by the oblique entrance of

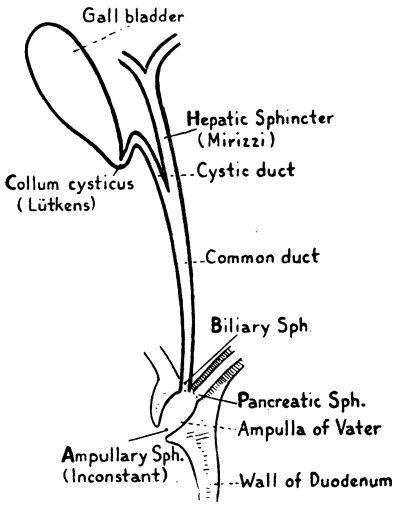


Fig. 1—Location of the sphincters of the biliary and pancreatic ducts.

the choledochus through the wall of the duodenum and by the folds of mucous membrane in the ampulla. The sphincter is most important in its relationship to the physiology of the gallbladder. When the sphincter is contracted between the periods of gastric digestion the normal gallbladder through its absorptive ability and elasticity concentrates and stores large amounts of bile which are continuously being secreted by the liver. The gallbladder acts as a pressure regulating apparatus and insures a continuation of bile secretion by preventing the intraductal pressure in the biliary tree from exceeding the secretory pressure of the liver. The intermittent discharge of bile into the intestine is dependent upon the relaxation of the sphincter mechanism and the simultaneous contraction of the intrinsic musculature of the gallbladder wall according to the Meltzer law of reciprocal innervation. The exact mechanism is not clear and has recently been questioned.[17] However, it is quite understandable that any gradual increase in the tonicity of the sphincter due to reflex causes may cause a stasis of bile, and due to the apparent lack of elasticity of the common bile duct may lead to dilatation of the gallbladder resulting in a hypotonic organ.[18] If, however, the gallbladder musculature responds with increased activity, a hypertonic type of gallbladder may develop. On the other hand, a sudden tonic spasm of the sphincter of Oddi which precipitates a

rapid increase in the intraductal pressure of the common bile duct, or a sudden distention of the gallbladder may cause an acute colic. Lütkens[19] described another sphincter located at the neck of the gallbladder, so-called collum cysticus, as being present in about 75 per cent of his cases. While his original observations were doubted by many, recent cholecystographic evidence affords positive proof of its existence.[10] Sudden spasm of this sphincter may, too, be another cause of gallbladder colic. Recently Mirizzi,[20] from cholangiographic studies, has described a physiologic sphincter at the level of the hepatic duct, a spasm of which may cause hepatic pain and transient jaundice. It becomes quite evident that spasm of any of these sphincters may result in an increase of intraductal pressure sufficient in intensity to produce colic. For this reason the removal of the gallbladder, especially the hypostatic, noncalculous type, may give little clinical relief because the original source of the symptomatology may have been a spasm of the sphincter of the common bile duct or the ampulla. In other cases the removal of an apparently innocuous gallbladder together with the cystic duct is occasionally followed by a complete disappearance of the symptoms. The colic may have been due to a reflex spasm of the collum cysticus impeding the free emptying of an actively contracting gallbladder. It is also likely that the normal activity of the sphincter may be simultaneously altered when once acute or chronic pathologic changes in the gallbladder have interfered with its physiologic functions. Clinically it is not unusual to observe jaundice in acute cholecystitis with or without stones and yet at operation to find no organic obstructive cause for the icterus. This transient jaundice is probably due to an obstruction caused by a temporary spasm of the sphincter. Inasmuch as the physiologic activity of the gallbladder and the various sphincter mechanisms are so intimately correlated, cholecystectomy for cholecystitis with or without calculi will invariably be followed by alterations in the sphincter mechanism and in the pathologic physiology of the common bile duct. What these changes are is still a matter of controversy. Some believe that following the removal of the gallbladder the sphincter may lose its tone and the common bile duct may dilate. Puestow[21] predicates this mainly upon the low intraductal pressure following cholecystectomy and the relief of symptoms produced by operation. This viewpoint has not been generally accepted. Judd and Mann[22] have noted dilatation of the common bile duct after cholecys-

tectomy in animals. Colp, Doubilet and Gerber[23] have reported that following section of the sphincter in some cholecystectomized dogs there is a diminution in the caliber of the duct and a reduction in the intraductal pressure. Bergh and Layne,[11] McGowan, Butsch and Walters,[24] and Doubilet and Colp[25] have demonstrated clinically by manometric readings and lipiodol injections of the intubated choledochus that immediate postoperative resistance is offered by the sphincter and that marked spasm may be produced by pharmacologic agents. Moreover, any sudden distention of the intubated choledochus may initiate severe colic. In fact, in some cases reoperated for a calculous obstruction years after cholecystectomy had been performed, an irritable spastic sphincter was suspected because of the high manometric readings obtained from the intubated, dilated and infected choledochus. Definite hypertrophy of the muscles about the ampulla, even to the point of stenosis, has been observed following cholecystectomy by Bergh,[16] Westphal,[26] del Valle[27] and others. These experimental and clinical observations all tend to support the existence of spasm of the sphincter of Oddi and lend support to the current concept that biliary dyskinesia is an instrumental factor, not only in the causation of gallbladder disease, but in the production of the acute and uncertain symptomatology subsequent to cholecystectomy. The fact that many patients gain eventual relief and few suffer severely from the postcholecystectomy syndrome may be explained in part by the gradual physiologic adaptations of the sphincters and the extra-hepatic bile ducts to the loss of the gallbladder. For, as Ivy has so aptly suggested, there is no reason to assume that the sphincter mechanism reacts the same in all cases; a sphincter which may have been spastic in the immediate postoperative period may lose its spasticity for a time and then regain it later. This may account for the occurrence of colic during convalescence, and its eventual disappearance following appropriate medical treatment. The persistence of the postcholecystectomy syndrome in other cases may be due to dyskinesia of the sphincter of Oddi secondary to intraductal organic disturbances such as recurrent or residual cystic and choledochal calculosis, cholangitis, or stricture formation. But the activity of the sphincter mechanism may cause other complications in the presence of the ampullary sphincter. A contraction of this muscle converts the common bile duct and the duct of Wirsung into one canal so that, depending upon the relative secretory pressure, bile may flow into the

pancreatic duct or pancreatic ferments may mix with bile in the choledochus. The admixture of pancreatic juice with bile in the choledochus is fairly frequent. It may be perfectly innocuous. We[28] were able to demonstrate the presence of pancreatic ferments in about 28 per cent of a series of sixty cases in which specimens of bile were obtained from the intubated choledochus. In this particular series the pancreatic reflux was due solely to a spasm of the ampullar sphincter. The pathologic effects of this phenomenon have been proven by the experimental evidence and clinical investigations of Westphal, Popper, Wolfer and others.[26] The evidence is now quite clear that pancreatic reflux may cause either a nonperforative bile peritonitis or an acute chemical cholecystitis. In a series of fourteen consecutive cases of acute cholecystitis the gallbladder bile contained pancreatic ferments which probably were the agents responsible for the acute chemical inflammation. It is possible that some cases of chronic cholecystitis and chronic choledochitis with or without stones may be the result of an intermittent pancreatic reflux. It has been appreciated since Opie, in 1906, reported his classic case of a biliary calculus occluding the ampulla that a retrojection of infected bile may cause an edema or an acute pancreatitis. The pathogenesis of this condition has been fully described by Dragstedt and his coworkers.

Dyskinesia of the ampullary sphincter following cholecystectomy may play a significant and important role in the clinical picture of the postcholecystectomy syndrome. Our follow-up studies in those cases in which the presence of a pancreatic reflux was proven have disclosed no clinical evidence upon which changes in the common bile duct could be predicated. Whipple reported three cases of irreparable stricture of the choledochus in which he felt sure that the common bile duct had not been injured at the time of the primary cholecystectomy. When these patients had become deeply jaundiced requiring a second operation nothing was found but a shred of dense connective tissue extending from the duodenum to the portal fissure. He felt that such an extensive destruction of the common bile duct could only be explained by an activated pancreatic reflux. It is barely possible that those cases of chronic choledochitis described by the French surgeons in which the common bile duct resembles a pipe stem, and into which a fine probe could not be introduced, may be due to repeated pancreatic reflux rather than to biliary stagnation.

The effects of postcholecystectomy biliary reflux are known. El-

man[29] and others have called attention to recurrent attacks of acute pancreatitis and pancreatic edema following cholecystectomy and have demonstrated an elevation of the blood amylase during the acute phase. It is more than likely that biliary reflux as a result of sphincter spasm accounted for the acute attacks of pain in the left upper quadrant which left a distinct soreness in the twenty-four cholecystectomized patients already described. Two of these cases eventually developed an acute pancreatitis.

The facts which have been presented seem to show that a dyskinesia of the sphincter mechanism contributes materially to the production of certain types of gallbladder disease and acts as a dominant factor in the production of the postcholecystectomy syndrome. The medical treatment therefore in both conditions is essentially the same. It should always be tried before surgery is advised. Little new has been added to the well recognized therapy of biliary colic. It is still customary to give morphine in adequate doses. This gives relief by blocking the higher nerve centers, but it simultaneously increases the muscle spasm of the sphincter mechanism to such a degree that pain often continues when the effect of the opiate has worn off. Small hypodermic doses of morphine may be used as a therapeutic test to substantiate a diagnosis of dyskinesia. It will often cause a tonic spasm of an irritable sphincter with an increase in the intraductal pressure sufficient to cause an attack simulating gallbladder colic. In some of these instances nitroglycerine may afford relief. However, even if this test is positive it does not rule out intraductal pathologic lesions which we have seen may contribute to the picture either of gallbladder disease or the postcholecystectomy syndrome. Antispasmodics such as amylnitrate, glycerol trinitrate, or theophylline, and others should be ideal pharmacologic agents. Unfortunately they are not always successful in relaxing a spastic sphincter. Best and Barr[30] feel that although the action of atropine is not constant, it is occasionaly effective. They recommend that if a combination of morphine and atropine does not give relief in three hours additional morphine by hypodermic injection and nitroglycerine by mouth should be tried.

Each patient complaining of gallbladder colic should be thoroughly examined in order to determine the factor producing the biliary dyskinesia. It is important to identify and neutralize if possible the precipitating causes. Pavel[31] has emphasized the importance of the psyche

in the production of biliary dyskinesia. During periods of intense emotional excitement sphincter spasm may become so severe as to produce jaundice and pain. This nervous instability, especially in female patients, is frequently combined with irregularities in menstruation and with symptoms of menopause. In cases such as these, relief has been obtained by the administration of sedatives and proper hormonal therapy. A spastic colon, too, undoubtedly plays a major role in the reflex production of right upper quadrant symptoms. Lahey and Jordan[32] have reported that in a series of sixty-five cases, 44 per cent of the patients whose gallbladders could not be visualized with dye showed filling of the organ after a period devoted to the active treatment of their constipation. In the present series of patients, 57 per cent gave a history of constipation prior to operation. Some cases were improved following cholecystectomy but only 37 per cent were cured. The administration of bile salts in adequate amounts, the judicious use of mineral oil and magnesium sulfate and daily enemata of sodium carbonate often lessened the distressing right upper quadrant symptoms. Dyspepsia which was present in 67 per cent of our patients was relieved in 42 per cent of the cases. Efforts were made to ameliorate these distressing symptoms. Measures were prescribed to induce the frequent relaxation of the sphincter and to avoid periods of tonic contraction. Patients were advised to eat bland and non-irritating foods and to take small meals at frequent intervals, for periods of gastric digestion are followed by a discharge of bile into the duodenum. By this simple expedient, biliary stagnation may be prevented. It has also been shown by Doubilet and Colp[25] that when hydrochloric acid in high concentration reaches the duodenum the increased acidity intensifies the tonus of the sphincter. Therefore, if a test meal disclosed evidences of hyperchlorhydria, anacids were unhesitatingly prescribed to lower gastric acidity in an attempt to diminish sphincter tone. In some cases in which the symptoms of dyskinesia were severe, lavage of the duodenum with magnesium sulfate appeared to relax the sphincter. These therapeutic and dietary measures were found to be effective in relieving the postcholecystectomy syndrome in most patients. In about 8 per cent of the cases the symptoms persisted regardless of treatment. When the attacks became increasingly severe, or were complicated by the presence of biliary obstruction or cholangitis, surgery was advised.

Exploration was usually performed under spinal anesthesia, because

it afforded the most complete muscular relaxation. In these secondary operations it was quite usual to find the omentum, colon, pylorus and duodenum distorted and bound to the under surface of the liver. These firm and dense adhesions may have contributed in part to the symptomatology. The stomach and duodenum were always meticulously examined for the presence of peptic ulcer. The fact that patients have had a cholecystectomy does not eliminate the possibility that an ulcer may have been overlooked or may have developed in the interim. Following the lysis of adhesions the identification of the foramen of Winslow, the stump of the cystic duct, and the choledochus did not present unusual difficulties unless these structures had been partially obliterated by stricture. But any structure which was thought to be the duct was always first aspirated for the presence of bile. The specimen was subsequently examined microscopically, chemically and bacteriologically. The common duct was then opened and carefully probed, especially in its terminal portion, for it is here that impacted stones may be missed. Immediate cholangiography was not practiced. The pancreas, too, was carefully examined. Palpation was often misleading for the organ, while not enlarged, may be the seat of a chronic inflammation as proven by a punch biopsy. The operative findings in this series were found to be calculi in the stump of the cystic duct in two patients, and in the common duct in five patients; evidences of cholangitis were present in two cases and in two others an acute pancreatitis was found. In two patients a stricture of the papilla was encountered, and in seven cases no visible pathologic lesions were found which might account for the episodes of postcholecystectomy colic. The symptoms were therefore attributed to an intractable dyskinesia of the sphincter of Oddi.

Several surgical methods have been advocated to relieve sphincter spasm. Dilatation of the sphincter may be accomplished by passing graduated sounds through the common bile duct into the duodenum. It is a popular but not an entirely innocuous procedure. If the dilatation has been forceful and the sphincter mechanism has been unduly traumatized, an ascending anaerobic infection by Clostridium welchii may occur with fatal results. Biliary duodenal intubation has been recommended by Duval[33] and Walzel[34] to counteract the spastic sphincter. A T or straight tube is inserted via the common duct and the sphincter into the duodenum. This indwelling tube not only provides for the

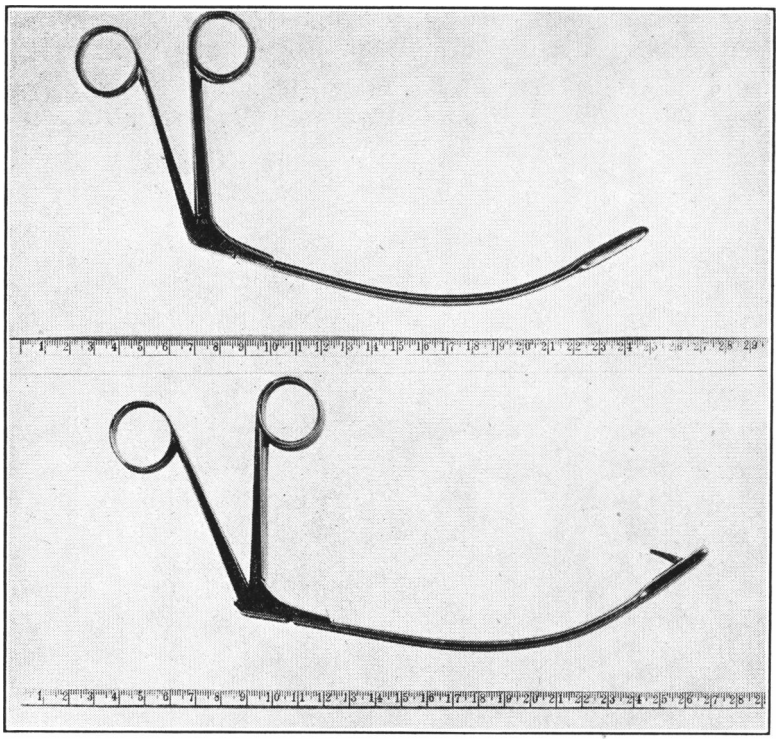


Fig. 2—The sphincterotome, closed and open.

immediate delivery of bile into the intestines but its mechanical presence maintains a continuous dilatation of the sphincter until the T tube is either withdrawn by the surgeon or the straight tube is eventually passed by the patient. Sasse,[35] Flörcken[36] and Petermann[37] have advised choledochoduodenostomy for either an obstructed or a spastic sphincter. An anastomosis between a dilated common bile duct and the contiguous duodenum certainly by-passes the sphincter mechanism. This procedure was proven to be most efficacious in the two cases of papillary stenosis in this series. However, choledochoduodenostomy, in addition to presenting certain technical difficulties, has another disadvantage. Theoretically, with the elimination of the sphincter mechanism there is the possibility of ascending infection by duodenal reflux. In spite of the fact that gastroduodenal roentgenograms in these patients often demonstrate the presence of barium or air in the common bile duct and its radicles, there is very little clinical evidence of cholangitis. The dilata-

tion of the sphincter, either by sounds or biliary duodenal intubation, is temporary and its effects are transitory. A dilated sphincter may eventually regain its tone and recurrent attacks of spasm may ensue. The result of choledochoduodenostomy is undoubtedly more permanent but the operative procedure has an appreciable mortality and a definite postoperative morbidity.

However, there is a direct surgical approach to the spastic sphincter. The sphincter of the common bile duct and sphincter of the ampulla if present may be paralyzed by surgical section of its fibers. This procedure has been done unwittingly since transduodenal choledochostomy was performed for impacted calculi, for it is impossible to extract the stones in these cases without first cutting the musculature of the sphincter mechanism. Archibald[38] was one of the first to divide the sphincter transduodenally for a dyskinesia. Del Valle[27] sectioned it through the duodenum for Odditis, a condition in which a retracted sphincter partially obstructed the flow of bile. Surgeons have been rather reluctant to deal directly with the sphincter in these unusual cases of intractable dyskinesia. It is quite difficult to identify the biliary papilla after the duodenum has been opened. But the papilla may be easily recognized if a large sized probe has first been introduced through the common bile duct into the region of the papilla. An incision through the anterior duodenal wall may then be made over this area. The sphincter musculature is easily divided under direct vision, following which the duodenostomy may be closed transversely. Recently Strode[39] described an effective and successful technique for transduodenal sphincterotomy in two cases of biliary dyskinesia. However, anterior duodenostomy may be followed by peritonitis, duodenal fistula and the physiologic disturbances due to duodenal adhesions. These complications may be obviated by endocholedochal sphincterotomy, a procedure in which the sphincter is divided by a specially designed instrument (Fig. 2) which has been introduced through the common bile duct. The experimental phase of this procedure has been described by Colp, Doubilet and Gerber[23] and the first case in which the operation was performed was reported in 1938.[40] Briefly, following a choledochostomy the patency of the papilla is first established by the passage of a fair sized probe into the duodenum. The closed sphincterotome is next introduced through the common bile duct and papilla into the duodenum. (Fig. 3). The sphincterotome is then opened, exposing the knife blade. The in-

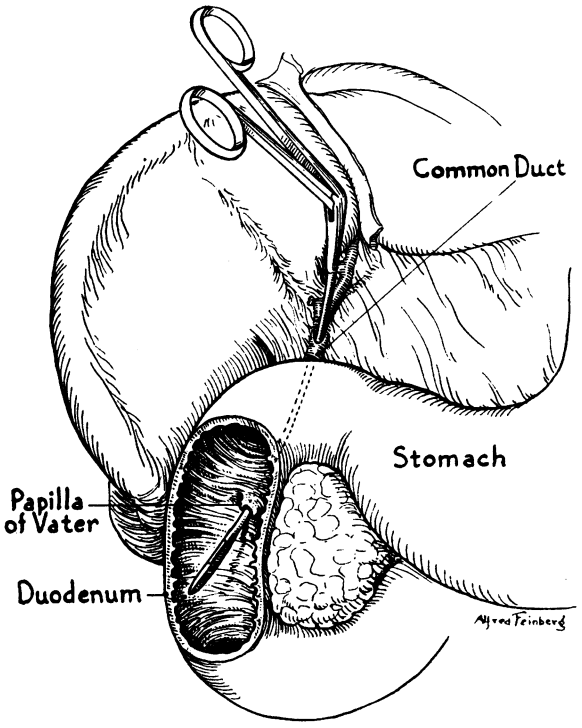
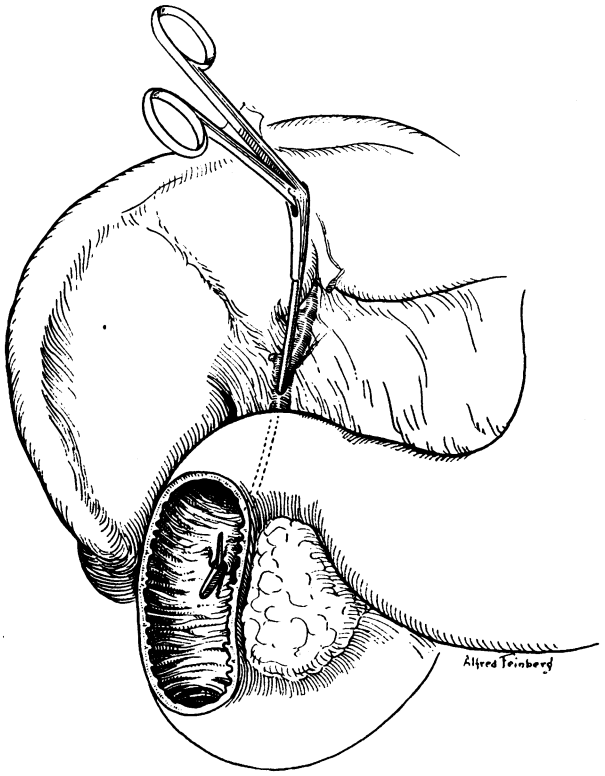


Fig. 3 — The closed sphincterotome is introduced through a cholecystectomy into the duodenum via the papilla of Vater.

strument is carefully withdrawn until the knife blade firmly impinges upon the sphincter. (Fig. 4). As the instrument is closed the knife is driven home, dividing the musculature, after which the sphincterotome may be easily withdrawn. The operation is simple and has not been followed by unusual bleeding. Often, upon opening the instrument, a specimen from the sphincter region may be found in the knife slot. The tissue thus obtained may be examined histologically. The common bile duct is then drained with a T tube. Drainage is important, not only because it provides an exit for bile if postoperative edema should develop in the region of the sphincter, but because it affords an opportunity for manometric readings and lipiodol studies. The drainage of the common bile duct is continued until the closure of the T tube is followed by the absence of pain and the presence of normal temperature. The postoperative course to date has been uneventful and no untoward sequelae developed which could possibly be attributed to the division of the



Fige. 4—The sphincterotome is opened and withdrawn until it firmly impinges upon the papilla of Vater.

sphincter. The number of cases in which this procedure has been done is still small. It has been used only in those patients in whom an intractable dyskinesia of the sphincter was apparently the sole cause for the postcholecystectomy syndrome. Its use has not been extended to paralyze an ampullary sphincter in those cases in which either a biliary or pancreatic reflux had been proven. There were seven cases in which endocholedochal sphincterotomy was performed. Six of the cases have been followed, one for six years, one for five years, one for three years, two for eighteen months, and one for six months. All have shown definite improvement and have been relieved of their troublesome symptoms. Whether surgical section of the sphincter permanently destroys its action, and whether the resulting scar will cause a secondary contraction of the papilla with stenosis and biliary stasis, only careful

follow-up of these and other cases will reveal. To date, none of these anticipated theoretical complications have resulted. Many of the other involuntary sphincters of man, when divided, never again fully regain their function, and when healing takes place a stenosis does not result. Perhaps the sphincter of the common bile duct and the ampulla may be placed in a similar category.

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